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L2 same (advantag$ or useful$)	9

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DATE: Wednesday, February 06, 2002 Printable Copy Create Case

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DB=USPT; PLUR=YES; OP=OR			
L3	L2 same (advantag$ or useful$)	9	L3
L2	L1 same antagonist$	42	L2
L1	peptide$ same (thrombin near0 receptor$)	127	L1

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FILE 'HOME' ENTERED AT 14:05:53 ON 06 FEB 2002)

FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE, REGISTRY' ENTERED AT 14:06:13 ON 06 FEB 2002

L1 2933 S PEPTIDE? (P)(THROMBIN(W)RECEPTOR?)
L2 406 S L1 (P)ANTAGONIST?
L3 35 S L2 (P)(ADVANTAG? OR USEFUL?)
L4 14 DUPLICATE REMOVE L3 (21 DUPLICATES REMOVED)

L3: Entry 1 of 9 File: USPT Sep 4, 2001

DOCUMENT-IDENTIFIER: US 6284871 B1
TITLE: Factor IX binding peptides, derived from factor VIII and their use as inhibitors of blood coagulation

Brief Summary Paragraph Right (8):
(d) Inhibitors of platelet activation and adhesion. Numerous studies have addressed this strategy, which has the theoretical advantage of specifically interrupting thrombin-dependent platelet recruitment at sites of vascular injury, while sparing the production of fibrin (see L. A. Harker et al., in: R. W. Colman et al. (Eds.), Hemostasis and Thrombosis, Basic Principles and Clinical Practice, 3rd edition, Lippincott, Philadelphia, 1994, pp 1638-1660). This strategy can be accomplished by various agents, including synthetic thrombin receptor antagonists, or monoclonal antibodies or peptides that interfere in the adhesion process. Although this approach seems particularly useful in arterial thrombosis, bleeding episodes still have been reported, suggesting that the specificity of this strategy may not have satisfactorily eliminated the antihaemostatic risk.

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L3: Entry 2 of 9 File: USPT Dec 5, 2000

DOCUMENT-IDENTIFIER: US 6156732 A
TITLE: Azole peptidomimetics as thrombin receptor antagonists

Brief Summary Paragraph Right (1):
Thrombin is an important serine protease in hemostatis and thrombosis. One of the key actions of thrombin is receptor activation. A functional human thrombin receptor, cloned by Coughlin in 1991 (T.-K. Vu, Cell 1991, 64, 1057), was found to be a member of the G-protein coupled receptor (GPCR) superfamily. The receptor activation putatively occurs by N-terminal recognition and proteolytic cleavage at the Arg-41/Ser-42 peptide bond to reveal a truncated N-terminus. This new receptor sequence, which has an SFLLRN [SEQ ID.:1] (Ser-Phe-Leu-Leu-Arg-Asn) N-terminus acting as a tethered ligand to recognize a site on the receptor, can trigger activation and signal transduction leading to platelet aggregation. Since 1991, two other protease-activated receptors with extensive homology to the thrombin receptor, "PAR-2" (S. Nystedt, Proc. Natl. Acac. Sci USA 1994, 91, 9208) and "PAR-3" (H. Ishihara, Nature 1997, 386, 502), were cloned, and found to be activated by similar N-terminal hexapeptide sequences. Thrombin receptor (PAR-1) specific antibody-induced blockade of the platelet thrombin receptor has shown efficacy against arterial thrombosis in vivo (J. J. Cook Circulation 1995, 91, 2961). Hence, antagonists of the thrombin receptor based on SFLLRN are useful in antagonizing these protease-activated receptors and as such may be used to treat platelet mediated thrombotic disorders such as myocardial infarction, stroke, restenosis, angina, atherosclerosis, and ischemic attacks by virtue of their ability to prevent platelet aggregation.

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L3: Entry 5 of 9 File: USPT Apr 6, 1999

DOCUMENT-IDENTIFIER: US 5892014 A
TITLE: DNA encoding a protease-activated receptor 3

Detailed Description Paragraph Right (83):
Specific receptor fragments of interest include any portion of the PAR3 which is capable of interacting with thrombin, for example, all or part of the extracellular domains predicted from the deduced amino acid sequence. Such fragments may be useful as antagonists (as described above), and are also useful as immunogens for producing antibodies which neutralize the activity of PAR3 in vivo (e.g., by interfering with the interaction between the receptor and thrombin). The sequence of FIG. 5B is most likely to bind thrombin. Modification of the (K38/T39) cleavage site for example, substitution of proline for T39 will render peptides mimicking this site uncleavable. Such peptides will bind thrombin with high affinity.

L3: Entry 8 of 9 File: USPT May 14, 1996

DOCUMENT-IDENTIFIER: US 5516889 A
TITLE: Synthetic thrombin receptor peptides

Detailed Description Paragraph Right (38):
The TRP linear and cyclic peptides and multimeric compounds of the present invention may be useful as agonist or antagonists in treating disorders where the thrombin receptor is involved, such as, but not limited to, cardiovascular disease, inflammatory disease, gastrointestinal disease, osteoporosis, tissue injury and repair (including nerve regeneration, both in the CNS and peripherally) and cancer in humans and the other animals.

Detailed Description Paragraph Right (40):
Thrombin receptor antagonists could prove of use in the setting of cardiovascular disease such as atherosclerosis and myocardial infarction from two perspectives. First, such antagonists will modulate platelet function and act synergistically with aspirin-related compounds that reduce platelet aggregation, thereby protecting against initial or repeat myocardial infarction, similar to the results obtained with other anti-platelet drugs. The thrombin receptor antagonists may also be useful in the setting of transient ischaemic attacks. Further, the anti-thrombin receptor drugs may be of use in the context of acute myocardial infarction, or other settings where a hypercoagulable state occurs. The thrombin receptor antagonist may be used to reduce thrombus formation and clot propagation either in the vicinity of damaged ventricular muscle or at the site of a pulmonary embolus. When present on solid supports, the agonists (or antagonists) may be used to attach to the surface of prostheses exposed to the blood to regulate the formation of clots or thrombi and to facilitate a healing process, thereby promoting the efficacy of an implanted prosthesis. Additionally, thrombin receptor antagonists may be used to retard the development of arterial plaque formation, wherein intimal vascular muscle hypertrophy and hyperplasia appears to play some role. The antagonist TRPs may be used in the setting of endarterectomy repair. The vascular endothelial cell, which is also thought to play a role in this process also is a target for the action of thrombin, and by extension, the receptor-related peptides. Since thrombin formation at such a site of vessel wall pathology likely plays a mitogenic role, the receptor antagonist would reverse such an action of thrombin both on the smooth muscle elements and on the endothelial cells.

Detailed Description Paragraph Right (42):
In the setting of tissue injury and repair, such as a wound or a surgical incision it could be advantageous to accelerate the healing process. The acceleration of would healing would also prove of value in the use of skin grafts for treating burn patients and in treatment of ocular injuries. It is likely that thrombin itself, generated at the site of injury, plays an important role in directing the healing process. In most settings, one would wish to accelerate the healing process; alternatively, in some situations, one might wish to delay the healing process to avoid the formation of inappropriate scarring, leading to the formation of cheloid. Thus, for sites of injury and tissue repair, it could prove of use to have available both the thrombin receptor agonists and thrombin receptor antagonists, depending on the situation. A non-degradable super-active analogue of the thrombin receptor peptide may be preferably used for such treatment in both humans and animals. The agents may also be used in the therapy of duodenal, ileal and colonic ulcer disease, as well as in the setting of nerve cell degeneration.

L3: Entry 3 of 9 File: USPT Jan 25, 2000

DOCUMENT-IDENTIFIER: US 6017890 A
TITLE: Azole peptidomimetics as thrombin receptor antagonists

Brief Summary Paragraph Right (1):
Thrombin is an important serine protease in hemostasis and thrombosis. One of the key actions of thrombin is receptor activation. A functional human thrombin receptor, cloned by Coughlin in 1991 (T.-K. Vu, Cell 1991, 64, 1057), was found to be a member of the G-protein coupled receptor (GPCR) superfamily. The receptor activation putatively occurs by N-terminal recognition and proteolytic cleavage at the Arg-41/Ser-42 peptide bond to reveal a truncated N-terminus. This new receptor sequence, which has an SFLLRN [SEQ. ID. NO:1] (Ser-Phe-Leu-Leu-Arg-Asn) N-terminus acting as a tethered ligand to recognize a site on the receptor, can trigger activation and signal transduction leading to platelet aggregation. Since 1991, two other protease-activated receptors with extensive homology to the thrombin receptor, "PAR-2" (S. Nystedt, Proc. Natl. Acac. Sci USA 1994, 91, 9208) and "PAR-3" (H. Ishihara, Nature 1997, 386, 502), were cloned, and found to be activated by similar N-terminal hexapeptide sequences. Thrombin receptor (PAR-1) specific antibody-induced blockade of the platelet thrombin receptor has shown efficacy against arterial thrombosis in vivo (J. J. Cook Circulation 1995, 91, 2961). Hence, antagonists of the thrombin receptor based on SFLLRN [SEQ. ID. NO:1] are useful in antagonizing these protease-activated receptors and as such may be used to treat platelet mediated thrombotic disorders such as myocardial infarction, stroke, restenosis, angina, atherosclerosis, and ischemic attacks by virtue of their ability to prevent platelet aggregation.